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DSM-IV personality disorders in the National Comorbidity Survey Replication

Mark F. Lenzenweger, PhD,
Department of Psychology, State University of New York at Binghamton

Michael C. Lane, MS,
Department of Health Care Policy, Harvard Medical School

Armand W. Loranger, PhD, and
Department of Psychiatry, Weill College of Medicine of Cornell University

Ronald C. Kessler, PhD
Department of Health Care Policy, Harvard Medical School

Abstract

BACKGROUND—The population prevalence of DSM-IV personality disorders (PDs) remains largely unknown. Data are reported here on the prevalence and correlates of clinician-diagnosed Clusters A, B, and C DSM-IV PDs in the general population of the US.

METHODS—PD screening questions from the *International Personality Disorder Examination* (IPDE) were administered in Part II (n = 5692) of the National Comorbidity Survey Replication (NCS-R). A probability sub-sample was then interviewed with the IPDE and used to link screening question responses with IDPE clinical diagnoses. The method of Multiple Imputation (MI) was then implemented to estimate prevalence and correlates of PDs in the full sample.

RESULTS—MI prevalence estimates were 5.7% Cluster A, 1.5% Cluster B, 6.0% Cluster C, and 9.1% any PD. All three PD clusters were significantly comorbid with a wide range of DSM-IV Axis I disorders. Significant associations of PDs with functional impairment were largely accounted for by Axis I comorbidity.

CONCLUSIONS—Strong Axis I comorbidity raises questions about the somewhat arbitrary separation of PDs from Axis I disorders in the DSM nomenclature. The impairment findings suggest that the main public health significance of PDs lies in their effects on Axis I disorders rather than in their effects on functioning.

Keywords

epidemiology; personality disorders; prevalence; comorbidity; mental health; National Comorbidity Survey Replication

Direct comments to Ronald C. Kessler, PhD, Department of Health Care Policy, Harvard Medical School, 180 Longwood Avenue, Boston, MA 02115 (e-mail: kessler@hcp.med.harvard.edu)

INTRODUCTION

The prevalence of personality disorders (PDs) in the general population of the United States has remained largely unknown ever since criteria for PDs were first published in the *DSM-III* (American Psychiatric Association 1980). Although various PDs, such as borderline PD, have been described as "common" (American Psychiatric Association 1987), such descriptions have been based on unsystematic samples (e.g., outpatient clinic patients, relatives of psychiatric patients) or clinical impressions (Weissman 1993). Three US studies have reported PD prevalence estimates based on rigorous clinical interviews in well-characterized non-patient samples (Crawford et al 2005;Lenzenweger et al 1997;Samuels et al 2002). (Table 1) The estimated point prevalence of any PD in these studies was in the range 9.0-15.7%. None of these studies, though, was broadly representative, making it impossible to generalize to the US as a whole. The current report addresses this problem by presenting the first nationally representative estimates of PD prevalence based on rigorous clinical interviews.

Prior research suggests that PDs are highly comorbid with a wide range of Axis I disorders (Goodwin et al 2005;Johnson et al 2005a;Loranger 1990), that the impairment in role functioning due to PDs is substantial (Johnson et al 2005b;Miller et al in press;Skodol et al 2002), and that people with PDs are heavy utilizers of both primary care and mental health services (Bender et al 2001;Miller et al in press;Moran et al 2001). Based on these observations, data are reported here not only on PD prevalence, but also on comorbidity between PDs and Axis I disorders, role impairments associated with PDs, and patterns of treatment among people with PDs.

METHODS

Sample

Data are based on the National Comorbidity Survey Replication (NCS-R), a nationally representative, face-to-face household survey of 9282 adults (ages 18+) in the continental US (Kessler and Merikangas 2004). The NCS-R was carried out by the professional field staff of the Survey Research Center at the Institute for Social Research, University of Michigan, between February 2001 and December 2003. Sampling was based on a multi-stage clustered area probability design. Informed consent was verbal rather than written to parallel procedures used in the baseline NCS (Kessler et al 1994). The response rate was 70.9%. Respondents received $50 for participation. A probability sub-sample of initial non-respondents was offered a higher financial incentive ($100) to complete a non-response survey. These procedures were approved by the Human Subjects Committees of Harvard Medical School and the University of Michigan.

All NCS-R respondents were administered a Part I diagnostic interview that assessed core disorders. A probability sub-sample of 5692 respondents, consisting of all Part I respondents who met criteria for a core disorder plus a roughly 25% probability sub-sample of other Part I respondents, was also administered a Part II interview that assessed disorders of secondary interest plus a wide range of correlates. The Part II sample is used in the current report. This sample was weighted to adjust for differential probabilities of selection within households and from the Part I sample, for differences in intensity of recruitment effort among hard-to-recruit cases, and for residual discrepancies with the 2000 Census on the cross-classification of socio-demographic and geographic variables. More complete information on the NCS-R sampling design and weighting procedures is reported elsewhere (Kessler et al 2004b).

The personality disorders screening questions

A series of PD screening questions from the *International Personality Disorder Examination* (IPDE) was included in the Part II NCS-R for each of the three PD Clusters A, B, and C, based on an analysis of a dataset from an earlier study (Lenzenweger 1999;Lenzenweger et al 1997). All screening questions found to be significant predictors of clinical diagnoses of any of these three classes of PDs or of any PD [including PD not otherwise specified (NOS)] based on the clinician-administered IPDE (Loranger 1999;Loranger et al 1994) in stepwise logistic regression analysis of the earlier dataset were included in the NCS-R. In addition, based on a special interest in two Cluster B PDs, antisocial personality disorder (ASPD) and borderline personality disorder (BPD), all IPDE screening questions for those two disorders were included in the NCS-R.

The clinical reappraisal interviews

Clinical reappraisal interviews with the IPDE were carried out with a probability sub-sample of 214 Part II respondents that over-sampled those who screened positive for one or more of our outcome measures based on the IPDE screening questions in the NCS-R. A veteran clinical interviewer (18 years of inpatient and outpatient psychiatric diagnostic experience) trained by the developer of the IPDE (AWL) and having extensive prior experience using the IPDE carried out all the clinical reappraisal interviews. The clinical interviewer was blind to the screening question responses. All clinical interviews were administered by telephone and were tape recorded for quality control. An experienced IPDE supervisor (MFL) monitored the tape recordings to prevent interviewer drift. Prior research has shown that the IPDE generates valid PD diagnoses when administered over the telephone (Rohde et al 1997). *DSM-IV* diagnoses based on the clinical interviews were generated for any Cluster A, any Cluster B, and any Cluster C PD as well as for ASPD, BPD, and any PD (including PD NOS). We note that the IPDE is commonly regarded as a conservative diagnostic instrument for the assessment of Axis II disorders relative to other available Axis II instruments. Moreover, we are not aware of any documented bias within the IPDE to generate differentially greater rates of positive diagnoses for any specific PD's.

Predicted probabilities of clinical diagnoses

Predicted probabilities of six IPDE diagnoses (Any Cluster A, Any Cluster B, Any Cluster C, Any PD including PD NOS, Antisocial PF, and Borderline PD) were assigned to each Part II NCS-R respondent who did not participate in the clinical reappraisal survey based on the results of stepwise logistic regression in the clinical reappraisal sample of clinical diagnoses on screening questions. No other individual PD diagnoses were assigned other than Antisocial and Borderline because the screening questions included in the full sample were too few in number to distinguish among these PDs with adequate precision. Only screening questions that were significant predictors at the .05 level were retained in the final models for the six outcomes. Prediction accuracy was excellent in all six equations, with area under the receiver operator characteristic curve (AUC), a prevalence-free measure of classification accuracy, of .88 for Cluster A, .99 for Cluster B, .80 for Cluster C, .93 for ASPD, .92 for BPD, and .91 for Any PD (including PD NOS).

Comorbid DSM-IV disorders

The Axis I *DSM-IV* disorders assessed in the core NCS-R/CIDI assessment include anxiety disorders (panic disorder with or without agoraphobia, generalized anxiety disorder, specific phobia, social phobia, agoraphobia without panic disorder, obsessive-compulsive disorder, post-traumatic stress disorder, separation anxiety disorder), mood disorders (major depressive disorder, bipolar disorder I or II or hypomania, dysthymic disorder), impulse-control disorders (oppositional-defiant disorder, conduct disorder, attention-deficit/hyperactivity disorder,

intermittent explosive disorder), and substance use disorders (alcohol and illicit drug abuse with or without dependence, nicotine dependence). Organic exclusion rules and diagnostic hierarchy rules were used in making diagnoses. We focus on 12-month prevalence of these disorders in the current report. As detailed elsewhere (Kessler et al 2004a;Kessler et al 2005a), blinded clinical reappraisal interviews using the Structured Clinical Interview for DSM-IV (First et al 2002) with a probability sub-sample of NCS-R respondents found generally good concordance between DSM-IV/CIDI diagnoses of anxiety, mood, and substance disorders and parallel diagnoses based on the SCID. Impulse-control disorder diagnoses were not validated.

Other correlates of personality disorders

We also examined three sets of other possible correlates: socio-demographics, role impairment, and 12-month treatment. Socio-demographics included gender, age at interview (18-29, 30-44, 45-59, 60+), race-ethnicity (Non-Hispanic White, Non-Hispanic Black, Hispanic, Other), completed years of education (less than high school graduation, high school graduation or GED, some college, college graduate), marital status (married or cohabitating, previously married, never married), and employment status (working, student, homemaker, other).

Role impairment was assessed with the World Health Organization Disability Assessment Schedule (WHO-DAS) (Chwastiak and Von Korff 2003), an instrument that evaluates functioning in three domains of basic activity (self-care, mobility, and cognition) and three domains of instrumental activity (days out of role, quality of productive role performance, quality of social role performance) over a 30-day recall period. Each of these six evaluations was assessed using a continuous scale with a theoretical range of 0-100. These scales were dichotomized for purposes of the present analysis to distinguish high impairment by selecting a cut-point as close as possible to the 80^{th} percentile of the distribution to define the roughly 20% of respondents with the highest impairment on the dimension.

Treatment was assessed by asking all Part II NCS-R respondents about past year treatment for any problems with emotions, nerves, or substance use by a psychiatrist, any other mental health professional (e.g., clinical psychologist, psychiatric social worker), a general medical health care provider, a human services professional (e.g., religious counselor, a social worker seen in at a social services agency), and in the complementary-alternative medicine (CAM) sector (either participation in a self-help group or treatment by a CAM professional).

Analysis methods

As noted above, the coefficients from best-fitting logistic regression equations of clinician PD diagnoses predicted by IPDE screening questions in the clinical reappraisal sample were used to assign a predicted probability of each PD diagnosis to Part II respondents who were not part of the clinical reappraisal sample. Further analysis was based on these predicted outcomes in the total Part II sample rather than on the clinical reappraisal sample only. We note, in this context, that the logistic regression equations used to assign a predicted probability of each PD to the Part II respondents that were not interviewed were derived from the nationally representative clinical reappraisal sample in this study rather than from the earlier study used to select the screening questions (Lenzenweger et al 1997).

The method of multiple imputation (MI) (Rubin 1987) was used to adjust estimates of coefficients and statistical significance in the Part II sample for the imprecision introduced by imputing clinical diagnoses rather than by carrying out clinical assessments for all respondents. MI was implemented in a four-part simulation that had the following features. First, ten pseudo-samples, each of size 214, were selected using stratified random sampling with replacement from the 214 clinical re-appraisal interviews. The final clinical PD prediction equations were

then re-estimated separately in each pseudo-sample. Second, ten sets of predicted probabilities, one for each pseudo-sample, were generated for each Part II respondent who did not participate in the clinical reappraisal survey. Third, these predicted probabilities were transformed to dichotomous case classifications by selecting independent random numbers from the binomial distribution for each imputation for each respondent. Fourth, the ten independent imputations were used to create ten separate datasets in each of which the substantive analyses were repeated. The parameter estimates in these ten replicated datasets (i.e., either prevalence estimates, estimates of regression coefficients, etc.) were averaged to obtain a best estimate of the parameters, while the MI variance of each averaged parameter estimate was obtained by combining the mean of the variance across the ten replications (i.e., the average within-replication variance) with the variance of the parameter estimate across the replications (i.e., the between-replication variance).

In considering the implications of the MI approach to imputation and variance estimation, it is important to recognize that prevalence estimates are unbiased to the extent that the clinical reappraisal sample is representative of the full sample, that estimates of correlates will be conservative to the extent that the predictors in the imputation equations fail to capture the effects of the substantive correlates, and that the between-replication variance will be small when, as in the current case, imputations are precise (i.e., AUC is high). This means that the precision of the parameter estimates will approach the precision that would have been achieved if PD had been directly measured rather than imputed in the total sample.

PD prevalence estimates were calculated as the means of the ten MI prevalence estimates. The proportion of respondents with the various PDs who received treatment in the past 12 months was also estimated as a mean of MI estimates. Associations of PDs with socio-demographics, measures of disability, and the Axis I DSM-IV disorders assessed in the NCS-R were estimated using logistic regression analysis, again with parameter estimates averaged over the ten MI replications. Logistic regression coefficients and their standard errors were exponentiated and are reported as odds-ratios (ORs) and their 95% confidence intervals (CIs). Because the NCS-R sample design features weighting and clustering, all parameter estimates were estimated using the design-based Taylor series linearization method (Wolter 1985) implemented in the SUDAAN software system (Research Triangle Institute 2002). Significance tests of sets of coefficients in the logistic regression equations were made using Wald χ^2 tests based on design-corrected MI coefficient variance-covariance matrices. Statistical significance was evaluated using two-sided design-based tests and the .05 level of significance.

RESULTS

The prevalence of DSM-IV personality disorders

MI prevalence estimates of IPDE/DSM-IV PDs in the total sample are 5.7%, 1.5%, and 6.0% for Clusters A through C, respectively. (Table 2) MI Prevalence estimates of BPD and ASPD are 1.4% and 0.6%, respectively. These estimates are all slightly lower than the direct estimates obtained in the clinical reappraisal sample. However, the latter are less accurate than the MI estimates due to the fact that the clinical reappraisal sample was only a small (n = 214) sub-sample of the full Part II NCS-R (n = 5692) and due to the fact that a somewhat higher proportion of IPDE screening questions were endorsed in the clinical reappraisal sample than the full sample. The MI estimates are also more precise than the clinical reappraisal sample estimates, as indicated by the consistently smaller standard errors of the former than the latter.

Prevalence estimates for individual PDs other than BPD and ASPD are available only in the clinical reappraisal sample. (Table 3) These estimates are likely to be somewhat higher than in the population because of the upward bias described in the last paragraph. Consistent with the cluster-level results, the highest prevalence estimates are in Cluster A (Schizoid 4.9%,

Schizotypal 3.3%, and Paranoid 2.3%) and Cluster C (Avoidant 5.2%, obsessive-compulsive 2.4%). It is important to note, though, that the rank ordering of prevalence estimates across individual PDs is imprecise due to the small size of the clinical reappraisal sample and the weighting and clustering of observations. This imprecision can be seen most clearly by examining the confidence interval of the PDs with the lowest prevalence estimates, histrionic and narcissistic PDs. Despite none of the clinical reappraisal sampling respondents meeting criteria for these PDs, an approximate estimate of the upper end of the 95% confidence interval of the prevalence estimate can be calculated based on information about sample size and the design effect of the other estimates (Hanley and Lippman-Hand 1983). This estimate is 1.3%, which is higher than the lower bound of the 95% confidence intervals of the prevalence estimates of all but one other individual PDs. The exception is avoidant PD. The upshot is that the only highly reliable difference in disorder-specific prevalence estimates in the clinical reappraisal sample is between the least prevalent (histrionic and narcissistic) and most prevalent (avoidant) PDs.

The sum of the prevalence estimates of all individual PDs in the clinical reappraisal sample (22.9%) is nearly twice as large as the prevalence estimate for any PD (11.9%), indicating that co-occurrence of PDs is a common occurrence. Tetrachoric correlations were calculated among all pairs of PDs to examine co-occurrence concretely. (Table 4) Average within-cluster correlations (.64-.74) are higher than average between-cluster correlations (.19-.30). Two-thirds of all correlations are significant at the .05 level, including 85% of within-cluster correlations and 62% of between-cluster correlations. All significant within-cluster correlations are positive, while 67% of between-cluster correlations are positive. All significant negative correlations involve either schizoid PD (with antisocial, dependent, and NOS) or dependent PD (with schizoid, schizotypal, antisocial, and NOS).

Socio-demographic correlates of DSM-IV personality disorders

Further analysis focused on the MI prevalence estimates in the total sample due to low statistical power to study correlates of directly estimated PDs in the clinical reappraisal sample. Socio-demographic correlates of the MI PD prevalence estimates are modest. (Results not presented, but available on request.) Sex, race-ethnicity, family income, and marital status are not significantly related to any of these PD measures, although there is a notable trend for ASPD to be less prevalent among women than men. Age and education are inversely related to Cluster B. Unemployment is positively related to BPD.

Comorbidity with DSM-IV Axis I disorders

The associations (ORs) of all six multiply imputed PD measures with the DSM-IV/CIDI Axis I disorders are consistently positive and substantial in magnitude, with a median OR of 6.0 and an inter-quartile range (IQR; 25^{th}-75^{th} percentiles among the ORs) of 5.2-7.6. (Table 5) The vast majority (88%) of these ORs are statistically significant at the .05 level. The ORs (with median and IQR reported in parentheses) are consistently much higher for Cluster B (8.3, 6.4-10.2) than for Cluster A (2.4, 2.1-2.5) or Cluster C (3.2, 2.6-4.1).

The range of ORs with Axis I disorders is quite narrow both for Cluster A and Cluster C, meaning that little differentiation can be seen across the Axis I disorders in the strength of association of these PDs. Considerably more differentiation exists in the ORs involving Cluster B, where the ORs are highest with dysthymic disorder, bipolar disorder, intermittent explosive disorder, and attention-deficit/hyperactivity disorder and lowest with specific phobia and nicotine dependence. It is noteworthy that the ORs associated with having any Axis I disorder are generally higher than those associated with specific disorders and that a strong relationship exists with number of Axis I disorders such that the OR with 3+ such disorders is consistently much higher than even the highest OR with any particular Axis I disorder.

These high ORs imply that large proportions of people with PDs also meet criteria for Axis I disorders. This is especially true for Cluster B. With regard to ASPD, 70.2% of cases also meet criteria for at least one 12-month Axis I disorder, with an average of 3.4 such disorders. (Table 6) With regard to BPD, 84.5% of cases meet criteria for one or more 12-month Axis I disorders, with a mean of 3.2. The proportions with Axis I comorbidity are lower, but still substantial, for Cluster A (41.1%) and Cluster C (49.7%), as are the mean number of Axis I disorders (2.2 for Cluster A and 2.5 for Cluster C). Given the comparatively low prevalence of PDs in relation to Axis I disorders, the proportions of Axis I cases with comorbid PDs are lower, but nonetheless nontrivial, with 25.2% of respondents who meet 12-month criteria for any Axis I disorder also meeting criteria for at least one PD.

The conditional prevalence of a PD is fairly similar for respondents with any anxiety disorder (30.0%), any mood disorder (38.1%), any impulse-control disorder (34.8%), and any substance use disorder (28.5%). All these estimates are higher than the conditional prevalence among respondents with any Axis I disorder because the latter include a higher proportion of people with only one disorder than do the sub-samples of respondents with specific classes of disorders. The conditional prevalence of having a PD is significantly lower among respondents with pure (single) Axis I disorders (14.6%) than with comorbid Axis I disorders (38.6%; $\chi^2_1 = 39.3$, $p < .001$).

Impairments in basic and instrumental functioning

Respondents with one or more PDs are significantly more likely than those without any PD to report high impairment in the three WHO-DAS areas of basic functioning (ORs in the range 2.4-5.1) as well as in the three WHO-DAS areas of instrumental functioning (3.1-7.4). (Table 7, Part I) The strongest association for each of the PDs is with high impairment in social role functioning. Cluster B is consistently associated with higher odds of impairment (3.1-11.7) than either Cluster A (1.1-2.5) or Cluster C (1.6-4.2).

The comorbidity of PDs with Axis I disorders raises the question whether Axis I comorbidity accounts for the high impairments associated with personality disorders. This possibility was evaluated by controlling four summary measures of Axis I comorbidity -- any anxiety, mood, impulse-control, and substance use disorders -- in evaluating the associations of PDs with the WHO-DAS measures. The ORs for the PDs became consistently much smaller when these controls were introduced. (Table 7, Part II) Compared to 80% significant in the absence of controls, only 17% of the PD ORs were significant after controlling for Axis I comorbidity, with a median (IQR in parentheses) OR in the presence of controls of 1.4 (1.3-1.6) compared to 3.2 (2.4-4.6) in the absence of controls. The effects of the Axis I disorders in predicting high impairment, in comparison, were consistently very powerful in all the prediction equations ($\chi^2_4 = 48.1 - 409.2$, $p < .001$). (Results not presented, but available on request.) Additional analyses failed to find significant interactions between PDs and Axis I disorders in predicting high impairment or stronger evidence of PD effects in the sub-sample of respondents with no Axis I disorders. (Results not presented, but available on request.)

Treatment

Thirty-nine percent (39.0%) of respondents with a PD reported receiving treatment for problems with their mental health or substance use at some time in the past 12 months. (Table 8, Part I) The percent in treatment was a good deal higher for Cluster B (49.1%) than either Cluster A (25.0%) or Cluster C (29.0%). The typical patient was seen in two treatment sectors, as indicated by the sum of treatment percentages across sectors being roughly twice the proportion of cases that received any treatment. A higher proportion of cases received treatment from general medical providers (19.0% overall, 13.5-26.0% across the different PDs) than from

either psychiatrists (14.3%, 10.0-21.7%) or other mental health professionals (17.3%, 9.5-23.5%), although the majority of cases in treatment were seen either by a psychiatrist or some other mental health professional (22.8%, 13.3-32.1%). Treatment was much less common in either the human services (8.2%, 3.8-10.7%) or CAM (7.6%, 3.2-13.4%) sectors. The proportion of respondents with PDs who were in treatment is significantly higher than that of demographically matched respondents without PDs. (Table 8, Part II) However, when we adjust for comorbid Axis I disorders, the associations between PDs and treatment become statistically insignificant. (Table 8, Part III)

DISCUSSION

The finding that roughly one-tenth of US adults suffer from a diagnosable personality disorder (including those with PD NOS) is broadly consistent with the three earlier US studies that, although based on less representative samples, used rigorous semi-structured clinical assessments to diagnose PDs in well-characterized non-patient samples. Similar results have been obtained in two European studies (Coid et al 2006;Torgersen et al 2001). A recent report based on a very large US national survey found a considerably higher prevalence of any PD despite omitting borderline, schizotypal, and narcissistic PDs (Grant et al 2004). This result must be viewed with caution, though, as it was based on a newly developed fully structured diagnostic interview carried out by lay interviewers rather than clinicians that lacked any accompanying validity data.

Estimates of relative prevalence of individual PDs in previous community studies based on rigorous clinical assessments are much less consistent than estimates of overall PD prevalence. Two of the three such studies found Cluster B to be more prevalent than Clusters A or C (Lenzenweger et al 1997;Samuels et al 2002), while the third found Cluster C to be more prevalent than Clusters A or B (Crawford et al 2005). Our study found Clusters A and C to be more prevalent than Cluster B. Specific differences across the studies with respect to each cluster are interesting to note. For the three prior US studies (Crawford et al 2005;Lenzenweger et al 1997;Samuels et al 2002), prevalence rates for Cluster A disorders ranged from 2.1% to 6.8% and our rate of 5.7% falls within this range. Similarly for Cluster C, the range across these three studies was 2.6% to 10.6%, and our rate of 6.0% for Cluster C accords well. Our rate for Cluster B disorders of 1.5% falls outside the range of 4.5% to 6.1% for the three prior studies. These discrepancies are not due to differences in diagnostic assessment, as three of the four studies used the IPDE to make diagnoses. Differences in sample composition are consequently the most plausible explanation for these discrepancies. For example, the Crawford et al. (2005) and Lenzenweger et al. (1997) study assessed subjects who were 22 and 18 years old respectively, whereas our study covered a broader age range and this may have impacted Cluster B rates as Cluster B disorders are more frequently diagnosed in younger people.

The rate of Cluster A disorders (5.7%) we found is interesting given the common clinical impression that such disorders are rare in hospital or clinic samples. Cluster A affected individuals may indeed be relatively rare in clinical samples owing to their tendency not to seek treatment. It is quite possible that the rate of Cluster A disorders is higher in the general population. Similar discrepancies have been noted for some Axis I disorders, such as pure generalized anxiety disorder and agoraphobia without panic disorder, both of which are much less common in comparative perspective in clinical than community samples due to treatment selection bias. In the same vein, our prevalence rate for Cluster B disorders (1.5%) might strike some as somewhat low in light of impressions based on clinic samples, however Cluster B disorders might actually be overrepresented in clinic samples owing to their tendency to display striking and clinically salient symptomatology (e.g., suicidal attempts, self mutilation,

aggression and impulsive dyscontrol). Cluster B disorders may quite possibly have a lower rate in the general population, particularly when a wide age range is sampled.

Little previous research has examined socio-demographic correlates of PDs, making it difficult to place in perspective our finding of weak socio-demographic correlates. The finding that young and poorly educated people have the highest prevalence of Cluster B PDs is generally consistent with the results of previous research on ASPD (Bland et al 1988;Meyers et al 1984;Morizot and Le Blanc 2003;Pevalin et al 2003) and BPD (Samuels et al 2002;Zimmerman and Coryell 1989), although our failure to find age differences in other PDs is inconsistent with the strong inverse association typically found in clinical samples (Mattia and Zimmerman 2001;Zimmerman and Coryell 1989). The DSM-IV (American Psychiatric Association 1994) suggests that borderline, histrionic and dependent PDs are more prevalent among women than men and that schizoid, schizotypal, narcissistic, paranoid, antisocial and obsessive-compulsive PDs are more prevalent among men than women (Corbitt and Widiger 1995). Such differences are generally found in clinical studies, especially with regard to antisocial, borderline, and dependent PDs (Corbitt and Widiger 1995;Loranger 1996;Reich 1987). Absence of significant sex differences in the NCS-R is consequently striking. These discrepancies could be due to ascertainment bias, base rate differences, or systematic differences in help-seeking related to socio-demographic factors in the clinical samples (Corbitt and Widiger 1995;Loranger 1996). For example, we did not find a sex difference in the rate of BPD in our study, whereas many clinical samples have found the diagnosis of BPD to be increased in women versus men. It may be, however, that the sex differences observed for the rate of BPD in clinical samples may actually reflect different base rates of men and women in such samples (e.g., Corbitt and Widiger 1995). We note Torgersen et al. (2001) and Zimmerman and Coryell (1989) did not find a sex difference for the rate of BPD in their large-scale nonclinical population studies as well. In sum, our data serve to extend and, perhaps, amend clinical impressions regarding the presumed relations of the PDs with various sociodemographic correlates by utilizing a large, randomly ascertained non-clinical population sample.

The finding that PDs are strongly comorbid with a wide range of Axis I disorders is broadly consistent with the results of previous, mostly clinical, studies (Dahl 1986;Dolan-Sewell et al 2001;Goodwin et al 2005;Johnson et al 2005a;Koenigsberg et al 1985;Loranger 1990;McGlashan et al 2000;Oldham et al 1995;Tyrer et al 1997;Zimmerman and Coryell 1989;Zimmerman et al 2005). Consistent with the NCS-R results, all forms of Axis I disorder have been found to be associated with higher levels of all three DSM PD clusters in these earlier studies. The NCS-R finding that little differentiation exists in the strength of comorbidity across different Axis I disorders for any given PD is also broadly consistent with the results of earlier studies, although it is important to note that many earlier studies focused on only selected Axis I disorders or evaluated PDs in samples of patients with Axis I disorders that had unusual features (e.g., among patients with panic disorder and suicidal behavior). However, the NCS-R results are inconsistent with the results of a comprehensive literature review that found Cluster B PDs to be more often comorbid with substance use disorders and major depression than with other Axis I disorders (Dolan-Sewell et al 2001). Given the evidence of good validity of the NCS-R assessments of both PDs and Axis I disorders, this discrepancy with earlier studies is likely to be due more to differences in sample composition than to differences in measurement.

It is noteworthy that the odds-ratios found in the NCS-R between PDs and Axis I disorders are comparable in magnitude to the odds-ratios found in separate NCS-R analyses between pairs of Axis I disorders (Kessler et al 2005b). This observation raises the possibility that PDs reflect variants on processes common to Axis I disorders and that PDs have been somewhat arbitrarily separated from Axis I disorders in the DSM nomenclature (Siever and Davis 1991;Widiger

2003). This is an important difference between the DSM and ICD systems, as the latter does not treat PDs as a separate Axis from other disorders (World Health Organization 1992). Our finding that comorbidity is much higher for Cluster B than Clusters A or C is an interesting variant on this theme. One possible explanation for this difference is that the dysregulation in underlying negative affect and constraint systems that affects the erratic and impulsive symptoms of Cluster B PDs (Depue and Lenzenweger 2001;Depue and Lenzenweger 2005) might be a more important determinant of Axis I disorders than of Clusters A or C PDs in the general population. That this specification has not been found in clinical studies could be due to a greater restriction in the variance of underlying dysregulation in clinical samples than the general population.

The finding that PDs are associated with a wide range of functional impairments is consistent with the definition of PDs in the DSM system as well as with the results of clinical studies (Casey et al 1985;Johnson et al 2005b;Miller et al in press;Skodol et al 2002). A similar result was documented in a longitudinal study of PDs in a community sample (Chen et al 2006). However, our results suggest that these associations are largely accounted for by comorbid Axis I disorders. The opposite pattern is generally found in clinical studies, where negative associations of personality disorders with role functioning are consistently documented among patients with Axis I disorders (Bender et al 2001;Connor et al 2002;Grilo et al 2005;Keel et al 2002).

A plausible interpretation of this discrepancy between the NCS-R results and the results of clinical studies is that functional impairment might influence help-seeking more strongly among patients with pure personality disorders than among those with Axis I disorders (mindful that such help-seeking may be prompted by spouses, other family members, or employers rather than by the patients themselves), while distress affects help-seeking more among patients with Axis I disorders than among those with pure personality disorders. If these differences in determinants of seeking treatment exist, they could lead to a bias in treatment samples for PDs to be associated with impairment independent of Axis I disorders even though this pattern is much weaker in the general population.

This possibility is indirectly consistent with our finding that help-seeking among people with PDs is strongly affected by Axis I comorbidity. Indeed, further analysis of these data (results available on request) showed that the effects of Axis I comorbidity in accounting for treatment among people with PDs is explained by the high role impairment associated with highly Axis I comorbidity. Importantly, Axis I disorders, unlike PDs, were found to continue to be associated with significantly elevated odds of treatment even after controlling for role impairment, presumably reflecting effects of clinically significant psychological distress on help-seeking. Finally, we note our findings regarding the relative impact of Axis I comorbidity on functional impairment alert us to the fact that prior findings on this issue derived from clinical samples may not generalize to the population.

The NCS-R results have to be interpreted in the context of the limitation that PDs were assessed comprehensively only in the sub-sample of respondents who received IPDE clinical reappraisal interviews. Clinical diagnoses of DSM-IV/IPDE PDs were imputed in the larger sample. Concern about this limitation is reduced by the fact that the AUC of the imputation equations was consistently quite high, which means that the imputed diagnoses are likely to be very similar to the diagnoses we would have obtained if full IPDE interviews had been administered to all respondents. In addition, the MI method adjusts for the imprecision in parameter estimates introduced by imputation. Prevalence is estimated without bias using MI, while MI estimates of associations involving PDs are conservative. The NCS-R finding that PD is a relatively common form of psychopathology can consequently be considered reliable, while the findings of high Axis I comorbidity and impairment can be considered conservative. Another potential

limitation concerns the possibility that the strength of association between PD screening questions and true diagnoses varies by respondent age, sex, or other variables examined as correlates of PDs. The clinical reappraisal sample was too small to investigate the possibility of such variation. To the extent that it exists, this variation would bias estimates of associations even though the prevalence estimates themselves are not biased. Future epidemiological research could address this problem either by including a clinical reappraisal sample large enough to estimate such interactions powerfully or by administering clinical interviews to the entire sample. A final limitation concerns the possibility that individuals with a PD might have declined to participate more often in this study and this would lead to an underestimation of PD prevalence rates. Although we did not address this directly, our data were weighted to account for an under-representation of those declining to participate due to Axis I disorders and given the comorbidity of Axis I and Axis II disorders, this methodological refinement may have helped to offset, in part, any tendency of PD affected persons to be non-responders. It remains conceivable, nonetheless, that our PD prevalence rates are somewhat underestimated.

The finding that Axis I comorbidity accounts for the impairment and help-seeking associated with PDs could be somewhat overstated due to the conservative bias in MI estimates of PD effects. However, this bias is likely to be small in light of the high AUC of the imputation equations. Based on this fact, it seems likely that PDs have only modest effects on functional impairment independent of Axis I disorders in the general population. Given the high comorbidity of PDs with Axis I disorders, though, and the especially high odds-ratios of PDs with high Axis I comorbidity, the possibility exists that PDs affect the onset, persistence, and severity of comorbid Axis I disorders. An investigation of this possibility was beyond the scope of our analysis because no information was collected in the NCS-R about age of onset or persistence of PDs in relation to age of onset and persistence of Axis I disorders. However, these results argue strongly that the investigation of PD effects on Axis I disorders should be a focus of future longitudinal epidemiological research on personality disorders.

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Table 1

Prevalence estimates of personality disorders in the three previous non-clinical US population studies that used validated structured interviews

	Study		
	Lenzenweger et al., 1997	**Samuels et al., 2002**	**Crawford et al., 2005**
Instrument	**IPDE***	**IPDE***	**SCID-II†**
Nomenclature	**DSM-III-R**	**DSM-IV**	**DSM-IV**
Location	**Ithaca, NY USA**	**Baltimore, MD USA**	**Upstate New York, USA**
Sample	**University students**	**Community sample**	**Community sample**
Cluster A			
Paranoid	1.0	0.7	5.1
Schizoid	1.0	0.9	1.7
Schizotypal	1.6	0.6	1.1
Cluster B			
Antisocial	0.6	4.1	1.2‡
Borderline	1.3	0.5	3.9
Histrionic	2.9	0.2	0.9‡
Narcissistic	2.7	0.0	2.2
Cluster C			
Avoidant	1.0	1.8	6.4
Dependent	0.6	0.1	0.8
Obsessive-Compulsive	1.3	…§	4.7
Passive-Aggressive	1.6	…§	…§
Any PD	11.0//	9.0	15.7
(n)	(1646/258)¶	(742)	(644)

*International Personality Disorder Examination (IPDE) DSM-III-R (Loranger et al 1994) and DSM-IV (Loranger 1999) versions

†Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II) (First et al 1994)

‡Antisocial PD and histrionic PD prevalence estimates were based on self-report data.

§Ellipses indicate not applicable

//Includes DSM-III-R sadistic PD as well as personality disorder “not otherwise specified” based on the IPDE

¶A two-stage case identification approach was used in which all 1,646 respondents were administered screening questions based on the IPDE and all screened positives plus a probability sample of screened negatives were administered the IPDE (n = 258).

Table 2

Multiply imputed prevalence estimates of DSM-IV/IPDE personality disorders in the Part II NCS-R (n=5692) compared to direct prevalence estimates in the PD clinical reappraisal sample (n = 214)

	Part II NCS-R		Clinical reappraisal sample	
	%	(se)	%	(se)
Cluster A	5.7	(1.6)	6.2	(2.2)
Cluster B	1.5	(0.3)	2.3	(0.8)
ASPD	0.6	(0.2)	1.0	(0.5)
BPD	1.4	(0.3)	1.6	(0.7)
Cluster C	6.0	(1.5)	6.8	(1.8)
Any[†]	9.1	(0.9)	11.9	(2.8)

*Significant at the .05 level, two-sided test

[†]Includes PD not otherwise specified

Table 3

Prevalence estimates of individual DSM-IV/IPDE personality disorders in the PD clinical reappraisal sample (n = 214)

	%	(se)	(95% CI)
Cluster A			
Paranoid	2.3	(0.8)	(0.7-4.0)
Schizoid	4.9	(2.2)	(0.6-9.2)
Schizotypal	3.3	(2.0)	(0.0-7.2)
Cluster B			
Antisocial	1.0	(0.5)	(0.0-1.9)
Borderline	1.6	(0.7)	(0.3-3.0)
Histrionic	0.0	(0.0)	(0.0-1.3)
Narcissistic	0.0	(0.0)	(0.0-1.3)
Cluster C			
Avoidant	5.2	(1.6)	(2.0-8.3)*
Dependent	0.6	(0.4)	(0.0-1.5)
Obsessive-compulsive	2.4	(0.8)	(0.8-4.0)
Other			
PD NOS	1.6	(0.7)	(0.3-2.9)

* Significant at the .05 level, two-sided test

Table 4

Tetrachoric correlations among DSM-IC/IPDE personality disorders in the PD clinical reappraisal sample (n = 214)

	NCSR IPDE Clinical Data: Tetrachoric correlation estimates										
	Cluster A				Cluster B			Cluster C			
	PAR	S'OID	S'TYP	ANY A	ANT	BOR	ANY B	AVO	DEP	OCD	ANY C
Cluster A											
Paranoid (PAR)											
Schizoid (S'OID)	.77*										
Schizotypal (S'TYP)	.48	.96*									
Any cluster A PD (ANY A)	--	--	--								
Cluster B											
Antisocial (ANT)	.73*	-.84*	.13	.56*	--						
Borderline (BOR)	.76*	.56*	.34	.58*	.64*						
Any cluster B PD (ANY B)	.83*	.46	.27	.65*	--	--	--				
Cluster C											
Avoidant (AVO)	.70*	.55*	.53*	.60*	.05	.54*	.44				
Dependent (DEP)	.20	-.84*	-.86*	.03	-.83*	.82*	.77*	.70*			
Obsessive-compulsive (OCD)	.59*	.40	.49	.49*	.45	.67*	.59*	.63*	.80*		
Any cluster C PD (ANY C)	.67*	.49*	.46*	.55*	.24	.55*	.45	--	--	--	
Total											
PD NOS (PD NOS)	.55	-.89*	-.10	.37	.90*	.55	.82*	-.27	-.79*	.64*	.43

* Significant at the .05 level, two-sided test

Table 5

Associations (odds-ratios) of multiply imputed DSM-II/IPDE personality disorders with 12-month DSM-IV/CIDI Axis I disorders[†] in the Part II NCS-R (n=5692)

	Cluster A		Cluster B						Cluster C		Any PD	
			Antisocial		Borderline		Any Cluster B					
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Anxiety												
GAD	2.3*	(1.4-3.6)	7.4*	(2.4-23.0)	6.9*	(3.0-16.0)	7.6*	(4.4-13.3)	3.4*	(2.1-5.7)	5.7*	(3.9-8.4)
Specific phobia	2.2*	(1.3-3.7)	2.0*	(0.6-7.2)	5.3*	(2.6-11.1)	4.5*	(2.3-9.1)	3.7*	(2.1-6.4)	4.5*	(3.1-6.7)
Social phobia	2.4*	(1.3-4.5)	4.3*	(1.4-12.8)	7.1*	(3.4-14.7)	6.1*	(2.8-13.0)	6.5*	(3.4-12.2)	9.9*	(6.3-15.5)
Panic disorder	3.1*	(1.5-6.5)	10.8*	(3.4-34.2)	10.0*	(4.1-24.6)	8.3*	(3.7-18.8)	4.2*	(2.3-7.6)	8.0*	(5.1-12.5)
Adult separation anxiety disorder	2.4*	(1.0-5.4)	6.8*	(1.6-29.3)	6.1*	(2.5-14.8)	8.3*	(3.6-19.2)	3.2*	(1.7-6.2)	6.6*	(2.6-17.1)
PTSD	2.5*	(1.4-4.6)	9.8*	(3.1-30.9)	5.8*	(2.8-11.9)	7.3*	(3.6-14.6)	3.2*	(1.8-5.6)	5.8*	(3.4-9.7)
Any anxiety	2.5*	(1.7-3.7)	5.4*	(2.3-12.5)	8.1*	(4.1-16.2)	8.4*	(4.8-14.6)	4.0*	(2.3-6.8)	7.0*	(4.6-10.8)
Mood												
Major Depressive Disorder	2.5*	(1.4-4.4)	3.2*	(0.8-13.2)	5.8*	(3.0-11.3)	4.3*	(2.2-8.6)	3.1*	(1.9-5.1)	6.1*	(3.7-9.9)
Dysthymia	2.5*	(1.3-5.1)	12.3*	(3.8-39.5)	12.1*	(5.5-26.6)	13.6*	(6.7-27.5)	4.2*	(2.3-7.5)	7.9*	(4.9-12.6)
Bipolar I or II	2.6*	(1.3-5.1)	11.2*	(3.3-37.8)	12.5*	(5.3-29.9)	11.3*	(5.4-23.7)	4.4*	(2.3-8.6)	9.8*	(5.1-18.8)
Any mood	2.6*	(1.6-4.1)	6.9*	(2.2-21.9)	9.2*	(5.0-16.8)	8.4*	(4.8-14.8)	3.6*	(2.3-5.7)	7.3*	(4.6-11.7)
Impulse-control												
Intermittent explosive disorder	2.7*	(1.4-5.0)	8.9*	(3.3-23.6)	12.5*	(4.3-36.0)	10.5*	(6.0-18.4)	2.5*	(1.3-4.8)	5.1*	(3.1-8.4)
Attention deficit disorder	2.0	(0.8-4.9)	8.9*	(3.3-24.5)	10.4*	(5.0-21.8)	9.8*	(5.5-17.5)	3.0*	(1.4-6.4)	6.5*	(3.9-11.1)
Any impulse	2.6*	(1.5-4.5)	8.3*	(3.0-22.9)	14.4*	(4.8-43.4)	11.3*	(6.5-19.8)	2.7*	(1.5-4.8)	5.9*	(3.7-9.2)
Substance												
Alcohol abuse or dependence	2.0	(0.9-4.5)	6.5*	(1.0-42.6)	11.3*	(5.6-22.8)	10.3*	(5.7-18.7)	1.9	(0.9-4.2)	4.7*	(2.7-8.1)
Drug abuse or dependence	1.6	(0.4-6.4)	7.2*	(1.6-31.8)	7.6*	(3.0-19.2)	8.7*	(3.4-22.1)	2.2	(0.7-7.3)	5.3*	(2.3-12.4)
Tobacco dependence	1.6	(0.6-4.3)	6.0*	(1.9-19.5)	3.7*	(1.7-8.2)	4.2*	(2.1-8.2)	1.8*	(1.0-3.1)	3.8*	(2.0-7.2)
Any substance	1.8*	(1.0-3.3)	7.2*	(2.2-24.4)	7.9*	(4.4-14.4)	8.1*	(5.0-13.0)	2.0*	(1.1-3.5)	4.7*	(2.6-8.5)
Overall												
Exactly 1	1.9*	(1.2-3.2)	3.9	(0.8-20.4)	7.1*	(1.5-33.2)	5.7*	(2.1-15.4)	2.4*	(1.4-4.2)	4.0*	(2.5-6.4)
Exactly 2	2.8*	(1.5-5.1)	8.6*	(1.9-38.5)	18.0*	(5.3-61.3)	14.5*	(5.4-39.2)	3.2*	(1.7-6.0)	7.9*	(4.3-14.3)
3 or more	3.5*	(2.0-6.2)	20.2*	(5.7-71.5)	48.7*	(10.3-230.1)	41.2*	(16.5-102.5)	6.7*	(3.3-13.9)	22.3*	(10.3-48.2)
Any of above	2.4*	(1.6-3.7)	8.5*	(2.7-26.3)	17.4*	(4.6-65.6)	14.4*	(6.1-34.0)	3.4*	(2.0-5.8)	7.4*	(4.5-12.5)

*Significant at the .05 level, two-sided test

†Based on logistic regressions of personality disorders on Axis I disorders controlling for socio-demographic variables

Table 6

Conditional prevalence of multiply imputed DSM-IV/IPDE personality disorders with 12-month DSM-IV/CIDI Axis I disorders in the Part II NCS-R (n=5692)

			Cluster B									
	Cluster A		Antisocial		Borderline		Any Cluster B		Cluster C		Any PD	
	Row* % (se)	Column* % (se)	Row* % (se)	Column* % (se)	Row* % (se)	Column* % (se)	Row* % (se)	Column* % (se)	Row* % (se)	Column* % (se)	Row* % (se)	Column* % (se)
Anxiety												
GAD	11.6 (4.3)	8.1 (1.6)	2.7 (1.0)	20.2 (8.5)	6.9 (2.5)	20.3 (5.5)	7.9 (1.9)	22.0 (4.6)	17.3 (4.0)	11.9 (2.3)	34.0 (3.8)	15.2 (1.8)
Specific phobia	11.0 (3.6)	14.1 (2.8)	1.0 (0.5)	12.5 (6.7)	5.7 (1.7)	30.3 (6.7)	5.3 (1.3)	26.7 (6.2)	16.9 (3.6)	21.1 (3.6)	28.8 (3.7)	23.4 (2.7)
Social phobia	12.1 (3.9)	11.4 (2.6)	2.0 (1.0)	19.5 (8.2)	7.3 (2.2)	28.4 (6.4)	7.2 (2.2)	26.4 (6.8)	25.2 (5.6)	22.8 (3.9)	44.5 (5.3)	26.3 (2.4)
Panic disorder	15.7 (6.4)	5.5 (1.5)	4.4 (1.8)	15.9 (6.8)	11.6 (4.1)	16.8 (4.7)	10.4 (3.1)	14.4 (4.3)	21.3 (5.2)	7.3 (1.7)	44.9 (5.7)	10.0 (1.4)
Adult separation anxiety disorder	12.8 (5.1)	4.3 (1.3)	3.9 (2.2)	13.0 (6.8)	8.9 (3.4)	12.4 (4.0)	12.4 (4.4)	15.9 (4.2)	16.7 (4.7)	5.4 (1.4)	42.5 (9.5)	8.9 (2.0)
PTSD	12.8 (5.0)	7.9 (1.8)	2.9 (1.2)	19.1 (8.3)	6.6 (2.2)	17.0 (4.5)	8.0 (2.3)	19.6 (5.5)	16.5 (3.9)	10.0 (2.1)	35.7 (4.6)	14.1 (2.1)
Any anxiety	10.7 (3.0)	31.0 (3.8)	1.6 (0.5)	47.5 (10.4)	5.0 (1.2)	60.5 (7.8)	5.5 (1.2)	61.4 (6.5)	14.9 (3.2)	41.4 (5.0)	28.8 (3.0)	52.4 (3.8)
Mood												
MDD	12.5 (4.1)	7.2 (1.7)	1.5 (0.8)	9.1 (5.7)	6.7 (2.2)	16.1 (4.1)	5.8 (1.8)	13.1 (3.6)	16.4 (4.1)	9.1 (1.9)	36.9 (5.4)	13.4 (1.9)
Dysthymia	13.2 (5.1)	5.3 (1.5)	4.4 (1.8)	18.4 (8.3)	11.3 (3.3)	19.0 (4.9)	13.1 (3.3)	20.8 (5.1)	20.8 (4.8)	8.1 (1.8)	42.7 (5.4)	10.8 (1.6)
Bipolar I or II	13.4 (5.0)	3.4 (1.0)	5.8 (2.7)	15.1 (6.6)	14.8 (5.1)	15.5 (4.5)	14.8 (4.4)	14.5 (3.6)	22.0 (6.9)	5.3 (1.4)	50.7 (7.5)	8.1 (1.3)
Any mood	12.4 (3.7)	12.4 (2.2)	2.7 (0.9)	27.7 (10.5)	8.2 (2.1)	34.3 (6.2)	8.4 (1.9)	33.0 (5.7)	17.4 (3.9)	16.8 (2.7)	38.1 (4.7)	24.1 (2.8)
Impulse-control												
IED	13.4 (4.5)	10.1 (2.7)	4.5 (1.7)	34.2 (9.4)	12.3 (4.3)	38.0 (10.3)	12.0 (3.1)	35.0 (6.2)	13.0 (3.6)	9.4 (2.3)	33.8 (5.8)	15.9 (2.4)
ADD	11.2 (5.4)	5.2 (1.8)	5.6 (2.3)	22.4 (8.3)	12.5 (4.2)	21.5 (5.4)	13.6 (4.0)	21.5 (4.7)	15.7 (4.9)	7.0 (2.1)	41.5 (6.3)	11.0 (1.7)
Any impulse	12.7 (4.2)	13.5 (3.1)	3.8 (1.3)	41.4 (10.5)	11.2 (3.6)	49.0 (11.1)	11.0 (2.7)	45.1 (6.3)	13.4 (3.3)	13.7 (2.8)	34.8 (5.0)	23.2 (2.8)
Substance												
Alcohol abuse or dependence	10.4 (3.7)	5.8 (2.2)	4.5 (2.6)	23.9 (11.6)	12.0 (3.7)	27.0 (6.8)	12.8 (3.8)	26.7 (5.6)	10.4 (3.5)	5.4 (1.8)	32.1 (5.7)	10.9 (2.1)
Drug abuse or dependence	9.0 (5.3)	2.3 (1.3)	5.5 (3.0)	13.6 (6.8)	10.8 (3.4)	11.1 (3.9)	13.7 (5.2)	12.9 (3.9)	11.5 (5.3)	2.7 (1.4)	36.7 (9.9)	5.6 (1.4)
Tobacco dependence	8.9 (4.1)	6.2 (2.1)	2.9 (1.2)	21.0 (8.5)	4.6 (1.4)	13.7 (4.4)	5.6 (1.6)	15.5 (3.9)	9.9 (2.6)	6.8 (1.6)	26.4 (5.1)	11.8 (2.2)
Any substance	9.4 (3.1)	11.9 (2.8)	3.2 (1.3)	40.5 (11.5)	7.2 (1.9)	38.2 (6.9)	8.2 (2.1)	39.9 (5.4)	10.2 (2.6)	12.4 (2.8)	28.5 (4.8)	22.6 (3.4)
Overall												
Exactly 1	8.0 (2.8)	19.0 (3.4)	0.8 (0.5)	20.1 (9.7)	2.0 (0.7)	19.5 (5.8)	2.0 (0.7)	18.3 (4.4)	8.9 (2.3)	20.5 (3.4)	14.7 (2.2)	22.2 (2.6)
Exactly 2	10.7 (2.9)	10.5 (2.2)	1.9 (1.1)	18.2 (9.0)	5.0 (1.4)	20.1 (5.5)	5.2 (1.5)	19.3 (5.1)	11.4 (3.8)	10.4 (1.9)	26.0 (4.0)	15.6 (1.9)
3 or more	13.7 (4.6)	12.8 (2.4)	3.7 (1.3)	35.2 (10.2)	11.6 (3.3)	44.9 (8.5)	12.5 (2.8)	45.4 (6.4)	21.3 (3.9)	19.4 (3.7)	49.5 (5.8)	29.2 (3.4)
Any of above	9.9 (2.9)	42.2 (4.8)	1.7 (0.5)	73.4 (10.2)	4.7 (1.1)	84.5 (8.3)	5.0 (1.0)	83.0 (5.7)	12.2 (2.6)	50.3 (5.2)	24.8 (2.7)	67.0 (4.3)

*Row percentages represent the percent of respondents with each of the Axis I disorders who meet criteria for the personality disorder. Column percentages represent the percent of respondents with each personality disorder who meet criteria for the Axis I disorder.

Table 7

Odds-ratios of high impairments in basic and instrumental role functioning among respondents with multiply imputed DSM-IV/IPDE personality disorders compared to other respondents without (Part I) and with (Part II) controls for comorbid Axis I disorders in the Part II NCS-R sample (n=5692)

	Cluster A		Cluster B						Cluster C		Any PD	
			Antisocial		Borderline		Any Cluster B					
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
I. Controlling for socio-demographics[†]												
Basic role functioning												
Mobility	1.7	(0.7-3.7)	3.7*	(1.3-10.6)	2.6*	(1.3-4.9)	4.0*	(2.1-7.6)	1.8*	(1.1-3.1)	2.7*	(1.9-3.9)
Self-care	1.1	(0.4-2.9)	3.3	(0.3-38.8)	2.6	(0.8-8.1)	3.1	(0.8-12.3)	1.6	(0.6-4.2)	2.4*	(1.3-4.4)
Cognition	2.3*	(1.1-4.8)	3.3	(0.8-13.9)	4.1*	(1.8-9.6)	4.8*	(1.7-13.4)	3.2*	(1.8-5.9)	5.1*	(3.2-8.2)
Instrumental role functioning												
Days out of role	1.7*	(1.1-2.7)	4.2*	(1.7-10.0)	4.3*	(2.2-8.5)	4.9*	(2.7-8.9)	2.0*	(1.4-3.0)	3.1*	(2.2-4.4)
	1.7*	(1.0-2.9)	4.9*	(1.9-12.7)	4.6*	(2.6-8.4)	5.1*	(2.9-9.0)	2.0*	(1.3-2.9)	3.2*	(2.3-4.6)
Productive role functioning												
Social role functioning	2.5*	(1.1-5.8)	5.6*	(1.2-26.4)	8.5*	(3.2-22.3)	11.7*	(5.1-27.1)	4.2*	(2.1-8.4)	7.4*	(4.4-12.5)
II. Controlling for socio-demographics and Axis I disorders[‡]												
Basic role functioning												
Mobility	1.3	(0.6-2.9)	1.6	(0.5-4.9)	0.9	(0.4-2.1)	1.6	(0.8-3.1)	1.3	(0.8-2.0)	1.5	(1.0-2.2)
Self-care	0.9	(0.4-2.1)	1.4	(0.1-17.6)	1.0	(0.3-3.4)	1.2	(0.3-5.6)	1.0	(0.4-2.6)	1.2	(0.7-2.3)
Cognition	1.4	(0.6-3.1)	0.8	(0.2-4.1)	0.8	(0.3-2.4)	1.0	(0.3-3.5)	1.5	(0.8-2.6)	1.7	(0.9-3.0)
Instrumental role functioning												
Days out of role	1.3	(0.8-2.0)	1.8	(0.6-5.4)	1.4	(0.6-3.5)	1.8	(0.9-3.5)	1.3	(0.9-2.0)	1.5*	(1.1-2.1)
	1.3	(0.7-2.2)	2.2	(0.7-6.2)	1.6	(0.8-3.3)	1.9	(1.0-3.5)	1.2	(0.8-1.9)	1.6*	(1.1-2.2)
Productive role functioning												
Social role functioning	1.3	(0.5-3.2)	1.4	(0.3-6.9)	1.6	(0.6-4.5)	2.7*	(1.1-6.5)	1.6	(0.9-3.0)	2.0*	(1.2-3.4)

*Significant at the .05 level, two-sided test

[†]Based on logistic regressions of functioning measures on personality disorders, controlling for socio-demographic variables

[‡]Based on logistic regressions of functioning measures on personality disorders, controlling for socio-demographic variables and for dichotomous measures of any 12-month anxiety disorder, any 12-month mood disorder, any 12-month impulse-control disorder, and any 12-month substance disorder

Table 8

Prevalence of 12-month treatment among respondents with multiply imputed DSM-IV/IPDE personality disorders† (Part I) and odds-ratios of treatment among respondents with multiply imputed DSM-IV/IPDE personality disorders compared to other respondents without (Part II) and with (Part III) controls for comorbid Axis I disorders in the Part II NCS-R sample (n=5692)

			Cluster B							
	Cluster A		Antisocial		Borderline		Any Cluster B		Cluster C		Any PD	
I. Treatment prevalence	%	(se)	%	(se)	%	(se)	%	(se)	%	(se)	%	(se)
Psychiatrist	7.8	(1.8)	13.0	(7.6)	21.7	(5.6)	20.1	(5.3)	10.0	(2.2)	14.3	(1.9)
Other mental health	9.5	(2.3)	23.5	(7.8)	22.1	(5.1)	23.5	(4.1)	12.4	(2.4)	17.3	(2.0)
Any mental health	13.3	(2.8)	28.2	(9.2)	29.4	(5.9)	32.1	(5.5)	16.7	(3.0)	22.8	(2.4)
General medical	13.5	(2.7)	20.2	(7.9)	21.8	(5.1)	26.0	(5.9)	14.3	(2.5)	19.0	(2.0)
Human service	3.8	(1.4)	9.3	(5.7)	8.5	(3.4)	10.7	(3.3)	6.1	(1.5)	8.2	(1.5)
CAM	3.2	(1.1)	13.4	(5.9)	9.8	(3.5)	13.4	(4.2)	5.2	(1.2)	7.6	(1.3)
Any	25.0	(3.8)	46.1	(10.0)	42.4	(6.0)	49.1	(6.5)	29.0	(3.9)	39.0	(3.3)
II. Unadjusted odds of treatment†	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Psychiatrist	1.9*	(1.1-3.4)	3.3	(0.8-13.9)	6.8*	(3.2-14.6)	5.9*	(2.8-12.4)	2.5*	(1.4-4.4)	4.7*	(3.0-7.3)
Other mental health	1.7	(0.9-3.2)	4.5*	(1.7-11.9)	4.2*	(2.2-8.2)	4.5*	(2.6-7.6)	2.2*	(1.3-3.7)	3.6*	(2.5-5.2)
Any mental health	1.7*	(1.0-3.0)	4.2*	(1.6-10.8)	4.6*	(2.4-8.5)	5.0*	(2.8-9.0)	2.2*	(1.3-3.7)	3.6*	(2.5-5.3)
General medical	1.7*	(1.0-2.8)	3.3*	(1.1-9.7)	3.3*	(1.5-7.2)	4.2*	(2.0-8.9)	1.7*	(1.0-2.7)	2.8*	(2.0-3.9)
Human service	1.1	(0.4-2.9)	2.5	(0.3-18.9)	2.4	(0.8-6.8)	3.1*	(1.4-7.0)	1.8*	(1.0-3.5)	2.7*	(1.6-4.5)
CAM	1.2	(0.5-2.7)	6.1*	(2.0-18.2)	4.0*	(1.7-9.5)	5.7*	(2.3-13.8)	2.0*	(1.1-3.5)	3.4*	(2.0-5.8)
Any	1.6*	(1.0-2.6)	4.6*	(2.0-10.6)	3.7*	(2.1-6.5)	4.9*	(2.7-8.8)	1.9*	(1.2-3.1)	3.5*	(2.4-5.0)
III. Adjusted odds of treatment‡	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Psychiatrist	1.2	(0.6-2.6)	0.6	(0.1-6.6)	1.6	(0.5-5.0)	1.4	(0.4-4.4)	1.2	(0.6-2.3)	1.8	(1.0-3.3)
Other mental health	1.2	(0.6-2.4)	1.6	(0.4-6.2)	1.2	(0.5-3.3)	1.4	(0.7-3.1)	1.2	(0.7-2.3)	1.7*	(1.1-2.7)
Any mental health	1.2	(0.6-2.4)	1.4	(0.3-6.4)	1.3	(0.5-3.3)	1.6	(0.7-3.8)	1.2	(0.7-2.2)	1.6	(1.0-2.6)
General medical	1.2	(0.7-2.0)	0.9	(0.1-5.4)	0.8	(0.3-2.3)	1.3	(0.5-3.5)	0.8	(0.5-1.4)	1.1	(0.6-1.9)
Human service	0.8	(0.3-2.3)	1.0	(0.1-11.3)	0.8	(0.2-3.1)	1.2	(0.4-3.5)	1.1	(0.6-2.3)	1.5	(0.8-2.9)
CAM	0.8	(0.4-2.1)	2.8	(0.8-9.8)	1.4	(0.4-4.6)	2.6	(0.8-8.0)	1.1	(0.6-2.2)	1.8	(0.9-3.4)
Any	1.1	(0.7-1.9)	1.8	(0.6-5.9)	0.9	(0.4-2.3)	1.5	(0.7-3.4)	1.1	(0.6-1.8)	1.5	(1.0-2.3)

*Significant at the .05 level, two-sided test

†Based on logistic regressions of treatment measures on personality disorders, controlling for socio-demographic variables

‡Based on logistic regressions of treatment measures on personality disorders, controlling for socio-demographic variables and for dichotomous measures of any 12-month anxiety disorder, any 12-month mood disorder, any 12-month impulse-control disorder, and any 12-month substance disorder